Sleep Medicine 15 (2014) 1259–1263

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Subjective sleep quality in stable neuromuscular patients under non-invasive ventilation

Grazia Crescimanno [a,b,*], Angela Misuraca [c], Giuseppina Purrazzella [c], Francesca Greco [c], Oreste Marrone [a]

[a] Italian National Research Council, Institute of Biomedicine and Molecular Immunology, Palermo, Italy
[b] Centre for Neuromuscular Disease, First Unit of Pneumology, V. Cervello Hospital, Palermo, Italy
[c] Italian Union Against Muscular Dystrophy (UILDM), Section of Palermo, Italy

ARTICLE INFO

Article history:
Received 20 February 2014
Received in revised form 7 May 2014
Accepted 5 June 2014
Available online 28 July 2014

Keywords:
Subjective sleep quality
Pittsburgh Sleep Quality Index
Neuromuscular disease
Non-invasive ventilation
Base excess

ABSTRACT

Objectives: Patients with neuromuscular diseases improve their sleep when treated with noninvasive ventilation (NIV), but their sleep architecture during NIV may still be disturbed by side effects of NIV or inadequacy of the ventilator setting. Little is known about subjective sleep quality during NIV. The aims of this study were to evaluate subjective sleep quality of stable neuromuscular patients under long-term NIV by using Pittsburgh questionnaire (PSQI), and to assess its possible determinants.
Methods: Fifty stable neuromuscular patients under long-term NIV were administered PSQI and underwent polysomnography. Arterial blood gases, forced vital capacity, and respiratory muscular strength were measured.
Results: Thirty-three patients had global PSQI ≥ 5 and were classified as bad sleepers. Good and poor sleepers differed in age ($P = 0.005$), base excess (BE) ($P = 0.02$), NIV inspiratory pressure ($P = 0.04$), %N1 ($P = 0.0006$), and %N3 sleep stage ($P = 0.02$). Percent N3 duration and Arousal/Awakening Index were correlated with rate of patient–ventilator asynchronies ($r = -0.41$ and 0.37, respectively, $P < 0.05$) and with NIV inspiratory pressure ($r = -0.44$ and 0.36, $P < 0.01$ and <0.05, respectively). At the final multivariate regression model, only BE and %N3 independently predicted global PSQI ($r^2 = 0.326$, $P < 0.001$).
Conclusions: Subjective sleep quality is often poor in neuromuscular patients under long-term NIV. Amount of slow wave sleep and chronic hypoventilation resulting in increased BE are independent predictors of subjective sleep quality. Since inadequate NIV setting or application can influence sleep structure and alveolar ventilation, great care should be paid to the setting and the correct application of NIV to ensure a better subjective sleep quality.

1. Introduction

Both subjective reports and sleep recordings have demonstrated that patients with neuromuscular disorders may suffer from a poor sleep quality [1]. Factors such as reduced spontaneous motility, pain or sleep respiratory disorders could contribute to impaired sleep quality [2,3]. Nocturnal non-invasive ventilation (NIV) is prescribed to most of these patients as their respiratory functional impairment progresses. Although this treatment improves nocturnal and diurnal gas exchange and sleep architecture, it does not always completely normalize them [4]. In fact, NIV itself could be responsible for some degree of discomfort [5]. Besides, in ventilated patients, patient–ventilator asynchronies (PVA) or persistent abnormal respiratory events (ARE), if present, can reduce sleep duration, increase the number of arousals [6,7], and could make subjective sleep quality worse.

Objective sleep quality during NIV has been often explored [4,7,8], but little is known about subjective quality of sleep in neuromuscular patients under NIV. Subjective and objective assessment of sleep quality may show some disagreement. In particular, in ventilated neuromuscular patients, we previously observed that subjective and objective estimates of sleep parameters are not always correlated, and in some cases may substantially differ [9].

Among possible tools to evaluate subjective sleep quality, the Pittsburgh Sleep Quality Index (PSQI) questionnaire is a well-validated one, and has been used in subjects with various kinds of disease, including neuromuscular illnesses [2]. In a recent paper in particular, sleep quality of ventilated neuromuscular patients evaluated by the PSQI is poor, but no correlates of this phenomenon were demonstrated [10]. A better knowledge of factors

* Corresponding author at: Istituto di Biomedicina e Immunologia Molecolare del C.N.R., Via Ugo La Malfa 153, 90146 Palermo, Italy. Tel.: +39 0916802615; fax: +39 0916890856.
E-mail address: grazia.crescimanno@ibim.cnr.it (G. Crescimanno).

http://dx.doi.org/10.1016/j.sleep.2014.06.010
1389-9457/

influencing subjective sleep quality could help us implement solutions to improve it.

The aim of this study was to assess subjective sleep quality by the PSQI and its possible determinants in neuromuscular patients under long-term nocturnal NIV in stable conditions. We analyzed the relationships of PSQI with the severity of diurnal respiratory dysfunction, characteristics of nocturnal respiratory function during NIV application, NIV setting parameters, and objective sleep quality assessed on polysomnographic EEG variables. We then investigated which of the explored variables could independently predict the PSQI score.

2. Methods

2.1. Setting and patients

This is a secondary analysis of a previous investigation designed to study the influence of home or hospital setting on polysomnography (PSG) outcomes and the reliability of self-reported sleep assessment during nocturnal NIV among patients with neuromuscular disease [9]. Fifty-two consecutive neuromuscular patients (34 with Duchenne muscular dystrophy, seven with congenital muscular dystrophy, four with mitochondrial myopathy, two with amyotrophic lateral sclerosis, two with spinal muscular atrophy, one with myasthenia gravis, one with congenital myasthenia, and one with facio-scapular-humeral dystrophy) in a stable condition, defined as absence of respiratory exacerbation during the previous 2 months, and who had been using NIV for more than 3 months, without any modification in ventilator setting, participated in this study. Twenty-seven patients were prescribed only nocturnal NIV application, whereas the other patients were requested to use NIV also for variable periods of the day. All patients were highly compliant to NIV (mean use: 11.2 ± 5.0 h/day). PSQI was administered to all patients. Patients then underwent PSG. The next morning, arterial blood gases determination, spirometry, and respiratory muscular strength assessment were performed. Anthropometrics, respiratory function, and PSG data are derived from the previous study [9]. No patient was assuming diuretics or corticosteroids at the time of this investigation.

2.2. Modes and settings of NIV

Methods adopted for ventilator setting have been already described [8]. Briefly, ventilator setting was established following both evaluations of diurnal comfort, respiratory function and gas exchanges, and of nocturnal in-hospital cardiorespiratory polygraphic monitoring. Forty-eight patients were treated with pressumetric and four with volumetric NIV. All patients used the same ventilator (IdeaUltra ResMed) with an optional double limb configuration incorporating an expiratory spirometer, and with an appropriate nasal or oronasal mask. The ventilator was equipped with built-in software (Easy diag Version 1.1.1, SAIME-RESMED, Savigny le Temple, France) for the recording and the measurement of several ventilation parameters, including mean nocturnal minute ventilation and leaks that were automatically calculated as percent differences between inspiratory and expiratory tidal volumes ($V_{Ti} - V_{Te}/100$).

2.3. PSQI questionnaire

PSQI is a self-administered questionnaire that assesses sleep quality and quantity over a month-long period [11]. The Italian version has been validated [12]. The PSQI was administered through an interview on the day preceding the sleep study. The questionnaire consists of 19 self-rated questions and of five questions that should be answered by bed-mates or roommates. The latter questions are used only for clinical information, but not in the scoring. The 19 questions are categorized into seven components, which are graded on a scale from 0 to 3. The PSQI components are as follows: subjective sleep quality (C1), sleep latency (C2), sleep duration (C3), habitual sleep efficiency (C4), sleep disturbances (C5), use of sleeping medication (C6), and daytime dysfunction (C7). The sum of scores for these seven components yields one global PSQI score that ranges from 0 to 21, where the highest score indicates the worst sleep quality. Subjects with a global PSQI score ≥5 are considered poor sleepers.

2.4. Polysomnography

PSG was performed either in hospital or at home, using the same device (SomnoLab 2 AASM, Weinmann, Hamburg, Germany) and methodology in both settings, and with the same technician engaged in setting up the recordings. Three unipolar EEGs (one frontal, one central and one occipital), right and left electro-oculograms, and electromyogram of the chin muscle for conventional sleep staging were recorded. Sleep and arousals were scored according to American Academy of Sleep Medicine 2007 criteria [13]. Total sleep time (TST), sleep efficiency (defined as TST/total recording time * 100), sleep onset latency (defined as time between lights off and first N1 stage sleep epoch), duration of each sleep stage as percent of the TST, and wake after sleep onset were calculated. Arousals lasting >15 s were classified as awakenings. An arousal/awakening index (AAI) was calculated as number of arousals + awakenings per hour of sleep time. Percentage of N3 and AAI were taken into consideration as indices of objective sleep quality. Analysis of respiratory function included scoring of ARE, classified according to Gonzalez-Bermejo et al. [14], of oxyhemoglobin saturation (SaO_2), and of PVA. The following SaO_2 parameters were calculated: mean SaO_2, lowest SaO_2, time spent with $SaO_2 < 90\%$, and oxygen desaturation index (number of oxygen desaturations ≥4% per hour). PVA were evaluated as previously described [8]. Patient–ventilator asynchrony index (PVA/I) was calculated as sum of all PVA events per hour of sleep time. Parallel to the PSG, transcutaneous CO_2 pressure ($PtcCO_2$) was recorded with a SenTec Digital Monitor (software version SMB SW-V04.03). The V-Sign™ Sensor was applied to the earlobe with a dedicated Ear Clip (SenTec AG, Therwil, Switzerland). The $PtcCO_2$ device was calibrated before and at the end of each recording to perform automatic drift correction, when necessary, and to improve interpretation of the $PtcCO_2$ values. From the $PtcCO_2$ recordings, mean $PtcCO_2$, peak $PtcCO_2$, and time spent with $PtcCO_2 > 45$ mmHg were automatically calculated after manual elimination of artifacts.

2.5. Respiratory function evaluation

Forced vital capacity (FVC) and respiratory muscle strength, assessed by maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP), were measured in the morning (Vmax22, Sensormedics, Yorba Linda, CA, USA). Arterial blood gases (ABGs) and acid–base balance were measured (ABG; BGE IL, Lexington, MA, USA) during spontaneous breathing except in five patients who were ventilator dependent.

2.6. Statistical analysis

Normally distributed data are presented as mean ± standard deviation (SD). Ordinal and continuous data that were not normally distributed, based on the Kolmogorov–Smirnov test, are presented as median and interquartile range. Square root transformation was used for non-normally distributed data. Differences in anthropometrics, pulmonary function, respiratory muscle strength, ABGs, polysomnographic, and ventilator data between good and poor sleepers were compared by unpaired *t*-test or Mann–Whitney *U*-test. Categorical variables were compared using the χ^2-test. Pearson's

Table 1
Diurnal respiratory function in good and poor sleepers.

	PSQI < 5 (n = 17)	PSQI ≥ 5 (n = 33)	P-value
FVC (L)	0.95 ± 0.5	0.90 ± 0.60	0.55
MIP (cmH_2O)	17.4 ± 13.0	22.2 ± 18.2	0.34
MEP (cmH_2O)	21.8 ± 11.7	21.5 ± 13.4	0.94
pH	7.40 ± 0.04	7.43 ± 0.03	0.053
HCO_3^- (mmo/L)	26.5 ± 2.7	28.1 ± 3.3	0.08
BE (mmo/L)	2.2 ± 2.4	4.0 ± 2.8	0.02
PaO_2 (mmHg)	92.3 ± 10	89.7 ± 9.3	0.34
$PaCO_2$ (mmHg)	41.6 ± 6.6	42.3 ± 5.8	0.79

PSQI, Pittsburgh Sleep Quality Index; FVC, forced vital capacity; MIP, maximal static mouth inspiratory pressure; MEP, maximal static mouth expiratory pressure; HCO_3^-, bicarbonates; BE, base excess; PaO_2, arterial oxygen pressure; PCO_2, arterial carbon dioxide pressure.
Data are expressed as mean ± standard deviation.

correlation analysis was used to assess the relationship of objective sleep parameters with respiratory and ventilation variables. To identify potential independent predictors of global PSQI, a stepwise backward linear regression model was used. $P < 0.05$ was considered significant. Statistical analysis was performed using a commercial software package (SPSS V. 19, Chicago, IL, USA).

3. Results

Two patients (both with myasthenia) were excluded due to incomplete data. All the remaining 50 patients were able to read, understand, and answer the questions and were included in the study. Mean global PSQI in these patients was 6.98 ± 3.2. Thirty-three/fifty patients (66%) had a global PSQI ≥ 5.

Good and poor sleepers differed in age (21.8 ± 6.6 vs 29.5 ± 9.8 years, respectively, $P = 0.005$), but not in BMI (22.1 ± 5.1 vs 21.0 ± 6.1 kg/m^2) or in gender distribution (female: 21% vs 22%). Diurnal and nocturnal respiratory function data are shown in Tables 1 and 2. On average, diurnal ABGs were normal in both groups. Base excess (BE) was higher in poor sleepers. FVC, MIP and MEP were severely impaired in both groups. Among both good and bad sleepers, subjects with normal and impaired nocturnal gas exchanges were included. Although BE was weakly correlated with lowest SaO_2 (r = –0.35, P = 0.012), time spent with SaO_2 < 90% (r = 0.28, P = 0.041) and $PtcCO_2$ > 45 mmHg (r = 0.28, P = 0.043), neither nocturnal SaO_2 nor $PtcCO_2$ values differed between groups. Similarly, no significant difference between groups was observed in PVA/I and ARE/h. In both groups residual ARE were rare (overall 50 and 92 ARE in good and bad sleepers, respectively) and included events with reduction in respiratory drive (54.0% and 57.6% of total ARE, respectively), events without reduction in respiratory drive and with upper airway obstruction (20.0% and 25.0%, respectively), and mixed events (26.0% and 17.4% respectively). Differences in NIV-related parameters are reported in Table 3. Inspiratory pressure (Pin) was lower in good than in bad sleepers, whereas back-up respiratory rate and air leaks recorded by the ventilators did not differ significantly between groups. Sleep structure in good and poor sleepers is shown in Table 4. Good sleepers showed a lower %N1 and a higher %N3 than bad sleepers.

In the whole patients' sample, at univariate analyses, neither %N3 nor AAI was correlated with BE. PVA/I and Pin were negatively correlated with %N3 (r = –0.41 and r = –0.44, P < 0.05 and P < 0.01, respectively). The same variables were positively correlated with AAI (r = 0.37 and 0.36, P < 0.05, respectively). In addition, AAI was positively correlated with ARE (r = 0.55, P < 0.01). Pin was not correlated with leaks or PVA/I.

At the final multivariate regression model, only BE and %N3 stage significantly and independently predicted PSQI (Table 5).

Table 2
Nocturnal respiratory polysomnographic variables in good and poor sleepers.

	PSQI < 5 (n = 17)	PSQI ≥ 5 (n = 33)	P-value
$PtcCO_{2peak}$ (mmHg)	46.4 ± 6.8	46.4 ± 5.9	0.60
$PtcCO_{2m}$ (mmHg)	40.3 ± 7.4	39.1 ± 6.6	0.55
$PtcCO_2$ > 45 (%)	0.2 (0.0–3.0)	0.0 (0.0–5.1)	0.93
ODI (no./h)	1.0 (0.1–3.4)	0.7 (0.2–2.2)	0.47
Lowest SaO_2 (%)	84.0 (79.7–88.0)	87.0 (83.0–91.0)	0.21
T < 90 (%)	0.0 (0.0–3.0)	0.0 (0.0–1.0)	0.11
SaO_{2m} (%)	97.6 ± 1.27	97.3 ± 1.6	0.44
ARE (no./h)	0.2 (0.0–0.5)	0.0 (0.0–0.6)	0.53
PVA/I (no./h)	11.2 (4.9–19.4)	14.0 (6.4–21.5)	0.32

PSQI, Pittsburgh Sleep Quality Index; $PtcCO_2$, transcutaneous CO_2 pressure; $PtcCO_{2peak}$, maximum $PtcCO_2$; $PtcCO_2$m, medium $PtcCO_2$; $PtcCO_2$ > 45, percentage of time with $PtcCO_2$ > 45 mmHg; ODI, number of oxygen desaturations ≥4% per hour of sleep; Lowest SaO_2, lowest arterial oxygen saturation; T < 90, % of time spent with SaO_2 < 90%; SaO_{2m}, mean nocturnal arterial oxygen saturation; ARE, abnormal respiratory events; PVA/I, patient–ventilator asynchrony index.
Data are expressed as mean ± standard deviation or median (interquartile range).

Table 3
Ventilator settings and nocturnal leaks in good and poor sleepers.

	PSQI < 5 (n = 17)	PSQI ≥ 5 (n = 33)	P-value
Mode of NIV, no. (%) patients			
Pressumetric	17 (100%)	29 (88%)	0.28
PACV	13 (76%)	20 (61%)	
PSV	4 (24%)	5 (15%)	
PSV-VG	0	3 (9%)	
Bi-PAP	0	1 (3%)	
Volumetric	0	4 (12%)	
Leaks (%)	46.76 ± 22.6	52.02 ± 16.9	0.35
Pin (cmH_2O)	16.0 ± 2.3	18.0 ± 3.3[a]	0.04
PEEP (cmH_2O)	4.4 ± 0.8[a]	4.6 ± 1.1[b]	0.66
RR (breaths/min)	14.6 ± 3.9	14.4 ± 2.8	0.82

PSQI, Pittsburgh Sleep Quality Index; NIV, non-invasive ventilation; PACV, pressure-assisted controlled ventilation; PSV, pressure support ventilation; ACV, assisted-controlled volume ventilation; PSV-VG, volume guaranteed pressure support ventilation; Bi-PAP, bi-level positive airway pressure; Pin, inspiratory pressure; PEEP, positive end-expiratory pressure; RR, respiratory rate.
Leaks were calculated as difference between volume delivered by the ventilator and expired tidal volume.
[a] Three patients only.
[b] Ten patients only.

4. Discussion

The results of this study show that in stable neuromuscular patients under long-term NIV, subjective sleep quality, assessed by PSQI, is often poor. Age, plasma BE, a higher Pin, and indices of poor objective sleep quality were individually associated with high global PSQI. Finally, only plasma alkalosis, as assessed by BE, and the

Table 4
Sleep structure in good and poor sleepers.

	PSQI < 5 (n = 7)	PSQI ≥ 5 (n = 33)	P-value
TST (min)	375.3 ± 82.5	344.5 ± 67.5	0.16
SE (%)	76.7 ± 14.4	70.0 ± 15.2	0.14
SOL (min)	24.7 ± 24.3	27.1 ± 33.0	0.79
WASO (min)	2.1 (1.4–21.7)	2.4 (1.3–11.1)	0.78
N1 (%)	11.9 ± 6.3	20.1 ± 11.4	0.006
N2 (%)	51.6 ± 6.9	49.6 ± 11.7	0.51
N3 (%)	20.1 ± 7.9	14.8 ± 7.7	0.02
R (%)	16.0 ± 6.9	15.0 ± 7.6	0.67
AAI (no./h)	20.3 ± 6.5	21.2 ± 9.34	0.70

PSQI, Pittsburgh Sleep Quality Index; TST, total sleep time; SE, sleep efficiency; SOL, sleep onset latency; WASO, wake time after sleep onset; N1, non-rapid eye movement stage 1; N2, NREM stage 2; N3, NREM stage 3; R, rapid eye movement stage; AAI, total number of arousals and awakenings/hour.
Data are presented as mean ± standard deviation or median (IQR).

Table 5
Relationship between PSQI and other selected variables estimated by stepwise multiple regression analysis.

Independent variables	B	SE	Beta	*t*	*P*-value
(Constant)	7.751				
BE (mmol/L)	0.552	0.144	0.450	3.837	0.000
N3 (% TST)	–0.162	0.048	–0.399	–3.401	0.001

PSQI, Global Pittsburgh Sleep Quality Index; SE, standard error; BE, base excess; N3, N3 sleep stage; TST, total sleep time.
Adjusted $R^2 = 0.326$; $F(2, 47) = 12.85$; $P < 0.001$.

duration of the N3 sleep stage were significant and independent predictors of the global PSQI.

Whereas a poor subjective sleep quality had already been reported in chronically ventilated neuromuscular patients [10], this study extends knowledge on this subject, showing how PSQI is related to objective sleep quality and which factors could contribute to make it worse. We observed multiple interrelationships among the PSQI, EEG, respiratory, and NIV variables.

To some extent, significant relationships between PSQI and sleep architecture might be expected. A previous paper showed that PSQI was significantly correlated with delta sleep duration and with microarousal index in healthy subjects who were about as young as our patients, unlike in middle-aged or elderly subjects [15]. Indeed, age may be an important source of disagreement between sleep quality evaluated with objective and subjective tools. Self-reported estimates may be vulnerable to memory processes, misperception, and overt or covert tendency to exaggerate number or gravity of symptoms, particularly in elderly patients. Patients enrolled in this study were young or middle-aged, which could make it easier to observe the relationship between the PSQI and objective sleep evaluation. However, not all aspects of subjective sleep evaluation may agree with objective assessment in ventilated neuromuscular subjects. In a previous paper we reported frequent discrepancies between subjectively evaluated and objectively measured parameters of sleep duration [9].

FCV, MIP, and MEP did not differ between good and bad sleepers. Unlike in patients who are not ventilated [16], in patients under NIV the severity of muscular respiratory impairment may not impact sleep, as mechanical ventilation can compensate for the effects of the respiratory muscles' weakness [17,18].

Nocturnal SaO_2 was not correlated to PSQI. Possibly, the narrow ranges of SaO_2 values in our patients' sample prevented detection of any significant difference between good and bad sleepers. BE was the only respiratory function variable measured during wakefulness that differed between the two groups. Recent papers have shown that alkalosis is associated with over-activity of principal cortical neurons, and that patients with alkalosis may manifest anxiety [19]. Additionally, there is evidence that PSQI is correlated with anxiety in the community as well as in some neuromuscular patients [20,21]. Thus, we hypothesize that patients with a high BE could be more anxious and perceive their sleep as less restorative in comparison with patients with a normal BE. In a study evaluating sleep quality by means of the Sickness Impact Profile questionnaire, Klang et al. observed no influence of diurnal blood gas values or bicarbonates in invasively and non-invasively ventilated subjects [22]; however, they did not report data on BE. Nardi et al. in neuromuscular patients under mechanical ventilation showed that BE may be a better hallmark of chronic hypoventilation than bicarbonates [23]. We found that BE, but not bicarbonates, significantly differed between poor and good sleepers: that could suggest that BE, which is a better marker of chronic hypoventilation, is more useful than bicarbonates when addressing subjective well-being in neuromuscular patients under NIV. Unlike the PSQI, sleep structure variables were not correlated with BE. BE reflects the average ventilation over several consecutive days and nights. Therefore, it could be more easily related to the PSQI, that is relevant to a 1-month period, than to sleep architecture recorded during a single night. Significant correlations between plasma alkalosis and sleep structure have been described in other conditions. In subjects sleeping at high altitude, alkalosis is associated with worse subjective and objective sleep quality, but this relationship is probably mediated by a greater amount of periodic breathing and respiratory disturbances, which is typical of sleep in that environment [24]. In patients recovering from acute respiratory failure, high pH and BE were correlated with less N3 and more arousals [25]: possibly, this relationship cannot be observed in patients in stable conditions such as those of this study.

Among variables relevant to mechanical ventilation, at univariate analyses Pin and PVA/I were correlated to objective sleep quality, as assessed by %N3 and AAI. Reasons why Pin was correlated to variables of sleep architecture are not clear. Increased leaks could mediate this relationship [26–28]. In addition, it has been demonstrated that a high Pin is associated with increased rate of PVA during wake [29]. However, among our patients Pin was not correlated either with leaks or with PVA/I. Possibly, higher Pin levels caused more discomfort due to gastric distension [30], which is common in neuromuscular patients with a high thoracic impedance, or required to apply the nasal/oronasal mask more tightly to prevent air leaks. The possible arousing effects of PVA are well known and may have been responsible for their significant correlation with AAI [31]. However, Pin and PVA/I were not shown to be independent predictors of global PSQI. Any effect they could exert on subjective sleep quality could probably be mediated by alterations in sleep architecture.

This study has some limitations. Our patients were affected by different neuromuscular diseases. However, when we evaluated Duchenne versus non-Duchenne patients, no differences were found in PSQI scores (data not shown). Besides, PSGs were performed in different environments, i.e. hospital or home, but we have already shown that there were no differences in terms of objective sleep quality in the different settings [9]. Finally, a first-night effect could have influenced the sleep structure in our patients. However, most patients did not complain of the PSG procedure and found it acceptable, as we previously reported [9].

In conclusion, nocturnal amount of slow wave sleep and chronic hypoventilation resulting in increased BE are independent predictors of global subjective sleep quality in stable neuromuscular patients chronically treated with NIV. According to these findings, great care should be paid to an effective and optimal NIV setting. In fact, inadequate NIV may be associated with insufficient nocturnal alveolar ventilation, which in turn may be responsible for plasma alkalosis. Besides, PVA or patients' discomfort due to excessive pressure support may affect sleep structure and, through this mechanism, subjective sleep quality. Therefore, quality and effectiveness of NIV may be critical to ensure a better subjective sleep experience in these subjects.

Funding

None.

Conflicts of interest

None declared.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.06.010.

References

[1] Piper AJ. Sleep and quality of life in neuromuscular disease. In: Verster JC, Pandi-Perumal SR, Streiner DL, editors. Sleep and quality of life in clinical medicine. Totowa, NJ: Humana Press; 2008. p. 209–20.

[2] Della Marca G, Frusciante R, Vollono C, Dittoni S, Galluzzi G, Buccarella C, et al. Sleep quality in facioscapulohumeral muscular dystrophy. J Neurol Sci 2007;263:49–53.
[3] Culebras A. Sleep and neuromuscular disorders. Neurol Clin 2005;4:1209–23.
[4] Contal O, Janssens JP, Dury M, Delguste P, Aubert G, Rodenstein D. Sleep in ventilatory failure in restrictive thoracic disorders. Effects of treatment with non invasive ventilation. Sleep Med 2011;12:373–7.
[5] Carron M, Freo U, BaHammam AS, Dellweg D, Guarracino F, Cosentini R, et al. Complications of non-invasive ventilation techniques: a comprehensive qualitative review of randomized trials. Br J Anaesth 2013;110:896–914.
[6] Rabec C, Rodenstein D, Leger P, Rouault S, Perrin C, Gonzalez-Bermejo J SomnoNIV group. Ventilator modes and settings during non-invasive ventilation: effects on respiratory events and implications for their identification. Thorax 2011;66:170–8.
[7] Fanfulla F, Taurino AE, Lupo ND, Trentin R, D'Ambrosio C, Nava S. Effect of sleep on patient/ventilator asynchrony in patients undergoing chronic non-invasive mechanical ventilation. Respir Med 2007;101:1702–7.
[8] Crescimanno G, Canino M, Marrone O. Asynchronies and sleep disruption in neuromuscular patients under home noninvasive ventilation. Respir Med 2012;106:1478–85.
[9] Crescimanno G, Greco F, Marrone O. Monitoring noninvasive ventilation in neuromuscular patients: feasibility of unattended home polysomnography and reliability of sleep diaries. Sleep Med 2014;15:336–41.
[10] Read J, Simonds A, Kinali M, Muntoni F, Garralda ME. Sleep and well-being in young men with neuromuscular disorders receiving non-invasive ventilation and their carers. Neuromuscul Disord 2010;20:458–63.
[11] Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193–213.
[12] Curcio G, Tempesta D, Scarlata S, Marzano C, Moroni F, Rossini PM, et al. Validity of the Italian version of the Pittsburgh Sleep Quality Index (PSQI). Neurol Sci 2013;34:511–19.
[13] Iber C, Ancoli-Israel S, Chesson A. Quan SF for the American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specification. Westchester: AASM; 2007.
[14] Gonzalez-Bermejo J, Perrin C, Janssens JP, Pépin JL, Mroue G, Leger P, et al. SomnoNIV Group. Proposal for a systematic analysis of polygraphy or polysomnography for identifying and scoring abnormal events occurring during non-invasive ventilation. Thorax 2012;67:546–52.
[15] Buysse DJ, Reynolds CF 3rd, Monk TH, Hoch CC, Yeager AL, Kupfer DJ. Quantification of subjective sleep quality in healthy elderly men and women using the Pittsburgh Sleep Quality Index (PSQI). Sleep 1991;14:331–8. Erratum in: Sleep 1992;15:83.
[16] Hukins CA, Hillman DR. Daytime predictors of sleep hypoventilation in Duchenne muscular dystrophy. Am J Respir Crit Care Med 2000;161:166–70.
[17] Toussaint M, Chatwin M, Soudon P. Mechanical ventilation in Duchenne patients with chronic respiratory insufficiency: clinical implications of 20 years published experience. Chron Respir Dis 2007;4:167–77.
[18] Toussaint M, Soudon P, Kinnear W. Effect of non-invasive ventilation on respiratory muscle loading and endurance in patients with Duchenne muscular dystrophy. Thorax 2008;63:430–4.
[19] Lu Y, Yi L, Liu D, Li J, Sun L, Zhang Z. Alkalosis leads to the over-activity of cortical principal neurons. Neurosci Lett 2012;525:117–22.
[20] Ramsawh HJ, Stein MB, Belik SL, Jacobi F, Sareen J. Relationship of anxiety disorders, sleep quality, and functional impairment in a community sample. J Psychiatr Res 2009;43:926–33.
[21] Leclair-Visonneau L, Magot A, Tremblay A, Bruneau X, Pereon Y. Anxiety is responsible for altered sleep quality in facio-scapulo-humeral muscular dystrophy (FSHD). Neuromusc Dis 2013;23:823–4.
[22] Klang B, Markström A, Sundell K, Barle H, Gillis-Haegerstrand C. Hypoventilation does not explain the impaired quality of sleep in post polio patients ventilated noninvasively vs. invasively. Scand J Caring Sci 2008;22:236–40.
[23] Nardi J, Prigent H, Garnier B, Lebargy F, Quera-Salva MA, Orlikowski D, et al. Efficiency of invasive mechanical ventilation during sleep in Duchenne muscular dystrophy. Sleep Med 2012;13:1056–65.
[24] Leaf DE, Goldfarb DS. Mechanisms of action of acetazolamide in the prophylaxis and treatment of acute mountain sickness. J Appl Physiol (1985) 2007;102:1313–22.
[25] Fanfulla F, Ceriana P, Artavilla D, Lupo N, Trentin R, Frigerio F, et al. Sleep disturbances in patients admitted to a step-down unit after ICU discharge: the role of mechanical ventilation. Sleep 2011;34:355–62.
[26] Meyer TJ, Pressman MR, Benditt J, McCool FD, Millman RP, Natarajan R, et al. Air leaking through the mouth during nocturnal nasal ventilation: effect on sleep quality. Sleep 1997;20:561–9.
[27] Teschler H, Stampa J, Ragette R, Konietzko N, Berthon-Jones M. Effect of mouth leak on effectiveness of nasal bilevel ventilatory assistance and sleep architecture. Eur Respir J 1999;14:1251–7.
[28] Bach JR, Robert D, Leger P, Langevin B. Sleep fragmentation in kyphoscoliotic individuals with alveolar hypoventilation treated by NIPPV. Chest 1995;107:1552–8.
[29] Carlucci A, Pisani L, Ceriana P, Malovini A, Nava S. Patient–ventilator asynchronies: may the respiratory mechanics play a role? Crit Care 2013;17:R54.
[30] Finder JD, Birnkrant D, Carl J, Farber HJ, Gozal D, Iannaccone ST, et al. American Thoracic Society. Respiratory care of the patient with Duchenne muscular dystrophy: ATS consensus statement. Am J Respir Crit Care Med 2004;170:456–65.
[31] Cabello B, Parthasarathy S, Mancebo J. Mechanical ventilation: let us minimize sleep disturbances. Curr Opin Crit Care 2007;13:20–6.